09/701014
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Term: L4 and neuro$ and pseudotyp$

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	DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=YES; OP=OR		
L7	L4 and neuro$ and pseudotyp$	8	L7
L6	L4 and neuro$	41	L6
L5	L4 and pseudotype$	18	L5
L4	rabies near10 G and retrovir$	78	L4
L3	L2 and neur$	0	L3
L2	6080912 [pn]	2	L2
L1	rabies near10 G and (neuronal or neuron$)	19	L1

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Search Results - Record(s) 1 through 41 of 41 returned.

☐ 1. 20030031681 . 13 Nov 01. 13 Feb 03. Combined growth factor-deleted and thymidine kinase-deleted vaccinia virus vector. McCart, J. Andrea, et al. 424/186.1; 435/235.1 435/456 A61K039/12 C12N015/863 C12N007/00.

☐ 2. 20020187951 . 08 Nov 01. 12 Dec 02. Lentiviral-mediated growth factor gene therapy for neurodegenerative diseases. Aebischer, Patrick, et al. 514/44; 424/93.2 A61K048/00.

☐ 3. 20020173049 . 14 Jun 01. 21 Nov 02. Controlling protein levels in eucaryotic organisms. Kenten, John H., et al. 436/501; 424/94.1 435/106 435/4 435/41 435/7.72 514/2 530/300 530/350 930/20 A01N037/18 C12Q001/00 C12P001/00 C12P013/04 C07K004/00 C07K007/00 C07K016/00 C07K001/00 A61K038/00 G01N033/53 A61K038/43 C07K002/00 C07K005/00 C07K014/00 C07K017/00 G01N033/566.

☐ 4. 20020168760 . 13 Mar 02. 14 Nov 02. Retroviral vectors for gene transfer into neuronal cells. Dornburg, Ralph C., et al. 435/320.1; 424/199.1 424/204.1 424/207.1 435/5 C12Q001/70 A61K039/12 A61K039/21 C12N015/00 C12N015/09 C12N015/63 C12N015/70 C12N015/74.

☐ 5. 20020150556 . 30 Mar 01. 17 Oct 02. Compositions and methods for tissue specific gene regulation therapy. Vile, Richard G., et al. 424/93.2; 435/320.1 435/366 435/456 A61K048/00 C12N015/86 C12N005/08.

☐ 6. 20020146843 . 14 Jun 01. 10 Oct 02. Controlling protein levels in eucaryotic organisms. Kenten, John H., et al. 436/501; 424/94.1 435/106 435/4 435/41 435/7.72 514/2 530/300 530/350 930/20 A01N037/18 C12Q001/00 C12P001/00 C12P013/04 C07K004/00 C07K007/00 C07K016/00 C07K001/00 A61K038/00 A61K038/43 C07K005/00 C07K017/00 G01N033/53 C07K014/00 C07K002/00 G01N033/566.

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☐ 8. 20020106729 . 29 Jun 01. 08 Aug 02. Expression vectors. Bleck, Gregory T.. 435/69.1; 435/320.1 435/325 435/456 435/6 536/23.2 C12P021/02 C12N005/06 C12N015/867 C12Q001/68 C07H021/04.

☐ 9. 20020042127 . 22 Nov 99. 11 Apr 02. DONOR CELLS EXPRESSING FUSOGENS. RUSSELL, STEPHEN JAMES, et al. 435/346; 435/449 A61K048/00 A01N063/00 C12N005/00 C12N005/06 C12N005/12 C12N015/02.

☐ 10. 20020019358 . 23 Apr 01. 14 Feb 02. Compositions and methods for in vivo delivery of polynucleotide-based therapeutics. Manthorpe, Marston, et al. 514/44; A61K031/70 A01N043/04.

☐ 11. 6495346 . 12 Jan 00; 17 Dec 02. Complex-forming proteins. Jerome; Valerie, et al. 435/69.7; 424/85.1 424/85.2 435/69.5 435/69.52 530/351 536/23.4 536/23.5 536/23.51. C12N015/62 A61K038/20 C07K014/54.

☐ 12. 6489142 . 25 Jan 01; 03 Dec 02. Methods and compositions for producing viral particles. Torrent; Christophe, et al. 435/69.1; 435/6 435/69.7 530/387.3 536/23.4 536/23.72. C12P021/06

C12P021/04 C12P021/08 C12Q001/68 C07H021/04.

☐ 13. 6468982 . 23 Jun 97; 22 Oct 02. Genetic immunization. Weiner; David B., et al. 514/44; 435/320.1 514/615 514/818 536/23.1. A61K048/00 A61K031/16 C12N015/74 C07H021/04.

☐ 14. 6348449 . 16 Dec 94; 19 Feb 02. Methods of inducing mucosal immunity. Weiner; David B., et al. 514/44; 424/130.1 424/184.1 424/209.1 435/235.1 435/252.3 435/320.1 435/455 514/2 514/330. A01N043/04 A61K031/70.

☐ 15. 6338953 . 11 Sep 97; 15 Jan 02. Expression of an exogenous gene in a mammalian cell by use of a non-mammalian DNA virus having an altered coat protein. Boyce; Frederick M., et al. 435/69.7; 435/252.3 435/320.1 435/325 435/69.1 514/12 536/23.2 536/23.5. C12P021/04 A61K038/00 C07H021/04 C12N015/00.

☐ 16. 6309647 . 15 Jul 99; 30 Oct 01. Poxvirus--canine dispemper virus (CDV) or measles virus recombinants and compositions and methods employing the recombinants. Paoletti; Enzo, et al. 424/199.1; 424/186.1 424/212.1 424/213.1 435/235.1 435/320.1 435/69.3 530/350. A61K039/275 A61K039/285 A61K039/165 A61K039/175 C12N007/01.

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☐ 23. 5997878 . 05 Jun 96; 07 Dec 99. Recombinant poxvirus-cytomegalovirus, compositions and uses. Paoletti; Enzo, et al. 424/199.1; 424/230.1 424/232.1 435/235.1 435/320.1 435/69.1 435/69.3. A61K039/245 A61K039/285 C12N015/00 C12N007/01.

☐ 24. 5981505 . 26 Nov 97; 09 Nov 99. Compositions and methods for delivery of genetic material. Weiner; David B., et al. 514/44; 424/278.1 514/615 514/818. A61K045/05 A61K048/00 A61K031/00.

☐ 25. 5962428 . 16 Sep 96; 05 Oct 99. Compositions and methods for delivery of genetic material. Carrano; Richard A., et al. 514/44; 424/278.1. A61K031/70 A61K045/05.

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☐ 27. 5911982 . 18 Apr 96; 15 Jun 99. Hz-1 virus persistence-associated-gene 1 (PAG1) promoter uses therefor, and compositions containing same or products therefrom. Chao; Yu-Chan. 424/93.2; 435/320.1 435/348 435/69.1 536/24.1. A61K031/70 C12P021/00 C12N005/06 C12N015/64.

☐ 28. 5863542 . 05 Apr 95; 26 Jan 99. Recombinant attenuated ALVAC canaryopox virus containing heterologous HIV or SIV inserts. Paoletti; Enzo, et al. 424/199.1; 424/188.1 424/208.1 424/232.1 435/236. A61K039/12 A61K039/21 A61K039/275.

☐ 29. 5843456 . 07 Jun 95; 01 Dec 98. Alvac poxvirus-rabies compositions and combination compositions and uses. Paoletti; Enzo, et al. 424/199.1; 424/201.1 424/202.1 424/204.1 424/205.1 424/218.1 424/224.1 435/235.1 435/252.3 435/320.1 435/69.3 514/2 530/350 530/826. A61K039/275 A61K039/295 A61K039/205.

☐ 30. 5833975 . 19 Jan 94; 10 Nov 98. Canarypox virus expressing cytokine and/or tumor-associated antigen DNA sequence. Paoletti; Enzo, et al. 424/93.2; 435/320.1 435/456 435/69.3 435/69.5 435/69.51 435/69.52. A61K048/00 C12N015/00 C12N015/86.

☐ 31. 5830876 . 30 May 95; 03 Nov 98. Genetic immunization. Weiner; David B., et al. 514/44; 424/278.1 514/615 514/818. A61K045/05 A61K048/00 A61K031/00.

☐ 32. 5817637 . 13 Jan 97; 06 Oct 98. Genetic immunization. Weiner; David B., et al. 514/44; 424/278.1 435/975 514/615 514/818. A61K045/05 A61K048/00 A61K031/00.

☐ 33. 5766599 . 01 Jun 95; 16 Jun 98. Trova fowl pox virus recombinants comprising heterologous inserts. Paoletti; Enzo, et al. 424/199.1; 424/232.1 435/235.1 435/320.1 435/69.1 435/69.3. A61K039/12 A61K039/275 C12N015/00 C12P021/06.

☐ 34. 5762938 . 21 Aug 96; 09 Jun 98. Modified recombinant vaccinia virus and expression vectors thereof. Paoletti; Enzo, et al. 424/199.1; 424/204.1 424/205.1 424/232.1 435/320.1 536/23.72. A61K039/12 A61K039/275 C12N015/00 C07H021/04.

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☐ 36. 5756102 . 06 Apr 94; 26 May 98. Poxvirus-canine distemper virus (CDV) recombinants and compositions and methods employing the recombinants. Paoletti; Enzo, et al. 424/199.1; 424/213.1 424/232.1 435/235.1 435/320.1 435/69.3. A61K039/175 A61K039/275 A61K039/285 C12N007/01.

☐ 37. 5739118 . 01 Apr 94; 14 Apr 98. Compositions and methods for delivery of genetic material. Carrano; Richard A., et al. 514/44; 424/184.1 424/278.1 435/375 435/69.1 435/69.3 514/25 514/27 514/33 514/35 514/510 514/54 514/680 514/731 514/732. A61K048/00 C07H015/24 C12N015/64 C12N015/87.

☐ 38. 5593972 . 21 Sep 93; 14 Jan 97. Genetic immunization. Weiner; David B., et al. 514/44; 424/278.1 514/615 514/818. A61K045/05 A61K048/00 A61K031/00.

☐ 39. 5494807 . 12 Aug 93; 27 Feb 96. NYVAC vaccinia virus recombinants comprising heterologous inserts. Paoletti; Enzo, et al. 435/69.3; 424/199.1 424/204.1 424/205.1 424/218.1 424/224.1 424/227.1 424/229.1 424/230.1 424/231.1 424/232.1 424/239.1 435/235.1 435/320.1 514/2 530/350 530/826. A61K039/285 A61K039/295 C12N007/01 C12N015/63.

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L4 and neuro$	41

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           15275  NEUROTROPIC?
      S1     235  RABIES AND NEUROTROPIC?
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Processing
Processed 10 of  35 files ...
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Processed 20 of  35 files ...
Completed processing all files
             235  S1
         4506392  G
         9469564  PROTEIN
          641955  GLYCOPROTEIN
          226619  G(N)(PROTEIN OR GLYCOPROTEIN)
      S2      36  S1 AND G (N) (PROTEIN OR GLYCOPROTEIN)
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>>>Unrecognizable Command
? rd s2
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      S3       9  RD S2 (unique items)
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13368892   BIOSIS NO.: 200100576041

Extensive attenuation of **rabies** virus by simultaneously modifying the dynein light chain binding site in the P protein and replacing Arg333 in the **G protein**.

AUTHOR: Mebatsion Teshome(a)
AUTHOR ADDRESS: (a)Department of Virology, Intervet International B.V., 5830 AA, Boxmeer: teshome.mebatsion@intervet.com**Netherlands

JOURNAL: Journal of Virology 75 (23):p11496-11502 December, 2001
MEDIUM: print
ISSN: 0022-538X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: **Rabies** virus (RV) is a highly **neurotropic** virus that

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migrates from the portal of entry to the central nervous system (CNS). The cytoplasmic dynein light chain (LC8), which is involved in a variety of intracellular motile events, was shown to interact with RV phosphoprotein (P). In order to determine the functional significance of this interaction, P residues 143 to 149 or 139 to 149 encompassing a conserved LC8-interacting motif (K/RXTQT) were deleted from recombinant viruses SAD-L16 and SAD-D29. These viruses are identical except for a replacement of the arginine at position 333 (R333) of the RV glycoprotein by an aspartic acid in SAD-D29. SAD-L16 virus is fully pathogenic for mice, whereas SAD-D29 is nonpathogenic for adult mice but retained pathogenicity for suckling mice. The deletions introduced into the LC8 binding site abolished the P-LC8 interaction and blocked LC8

incorporation into virions. All the mutants propagated in cell culture as efficiently as the parent strains. The pathogenicity of the mutants was then compared with that of the parent viruses by inoculating suckling mice. SAD-L16 derivatives were as pathogenic as their parent virus after intramuscular inoculation, indicating that LC8 is dispensable for the

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spread of a pathogenic RV from a peripheral site to the CNS. In contrast, SAD-D29-derived deletion mutants were attenuated by as much as 30-fold after intramuscular inoculation but remained as pathogenic as the parent virus when inoculated directly into the brain. This remarkable attenuation after intramuscular but not after intracranial inoculation suggested that abolishing the P-LC8 interaction reduces the efficiency of peripheral spread of the more attenuated SAD-D29 strain. These results demonstrate that elimination of the LC8 ligand and simultaneous substitution of R333 considerably attenuate RV pathogenicity and may be helpful in designing and developing highly safe live-RV-based vaccines.

DESCRIPTORS:
MAJOR CONCEPTS: Immune System (Chemical Coordination and Homeostasis); Infection; Pharmacology
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia; Rhabdoviridae (animal host only)--Animal Viruses, Viruses, Microorganisms

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ORGANISMS: mouse (Muridae)--host, suckling; **rabies** virus (Rhabdoviridae (animal host only))--SAD-D29, SAD-L16, extensive attenuation, pathogen
ORGANISMS: PARTS ETC: brain--nervous system; central nervous system {CNS }--nervous system
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animal Viruses; Animals; Chordates; Mammals; Microorganisms; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates; Viruses
CHEMICALS & BIOCHEMICALS: **G protein**; P protein--dynein light chain binding site; dynein light chain {LC8}; glycoprotein--P-LC8 interaction; live-**rabies** virus-based vaccine--development, immunostimulant-drug, safe
MISCELLANEOUS TERMS: vaccine development
CONCEPT CODES:
10064 Biochemical Studies-Proteins, Peptides and Amino Acids
12512 Pathology, General and Miscellaneous-Therapy (1971-)
20504 Nervous System-Physiology and Biochemistry

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22002 Pharmacology-General
22018 Pharmacology-Immunological Processes and Allergy
25502 Developmental Biology-Embryology-General and Descriptive
33506 Virology-Animal Host Viruses
34502 Immunology and Immunochemistry-General; Methods
36006 Medical and Clinical Microbiology-Virology

BIOSYSTEMATIC CODES:
02624 Rhabdoviridae-animal host only (1993-)
86375 Muridae

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DIALOG(R)File 5:Biosis Previews(R)

12688442 BIOSIS NO.: 200000441944
Reinvestigation of the role of the **rabies** virus glycoprotein in viral pathogenesis using a reverse genetics approach.
AUTHOR: Morimoto Kinjiro; Foley Heather D; McGettigan James P; Schnell Matthias J; Dietzschold Bernhard(a)
AUTHOR ADDRESS: (a)Center for Neurovirology, Department of Microbiology and Immunology, Thomas Jefferson University, 1020 Locust Street, Philadelphia, PA, 19107**USA
JOURNAL: Journal of Neurovirology 6 (5):p373-381 October, 2000
MEDIUM: print
ISSN: 1355-0284
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

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ABSTRACT: The **rabies** virus **glycoprotein** **(G)** gene of the highly neuroinvasive and **neurotropic** strains SHBRV-18, CVS-N2c, and CVS-B2c was introduced into the non-neuroinvasive and less **neurotropic** SN-10 strain to provide further insight into the role of G in the pathogenesis of **rabies**. Phenotypic analyses of the recombinant viruses revealed, as expected, that the neurotropism of a particular **rabies** virus strain was a function of its G. Nevertheless, the pathogenicity of the recombinant viruses was, in every case, markedly lower than that of the wild-type viruses suggesting that while the G dictates neurotropism, other viral attributes are also important in pathogenesis. The low pathogenicity of the recombinant viruses is at least in part due to a strong increase in transcription activity. On the other hand, the production of infectious virus by the R-SHB18 recombinant virus-infected cells was significantly delayed by comparison with SHBRV-18 wild-type virus infected-cells. Replacement of the R-SHB18 G cytoplasmic domain, transmembrane domain, and stem region with its SN-10 G counterparts neither results in a significant increase

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in budding efficiency nor an increase in pathogenicity. These results suggest that an optimal match of the cytoplasmic domain of G with the matrix protein may not be sufficient for maximal virus budding efficiency, which is evidently a major factor of virus pathogenicity. Our studies indicate that to maintain pathogenicity, the interactions between various structural elements of **rabies** virus must be highly conserved and the expression of viral proteins, in particular the **G protein**, must be strictly controlled.

DESCRIPTORS:
MAJOR CONCEPTS: Molecular Genetics (Biochemistry and Molecular Biophysics); Infection
BIOSYSTEMATIC NAMES: Animalia; Cricetidae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia; Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia; Rhabdoviridae (animal host only)--Animal Viruses, Viruses, Microorganisms
ORGANISMS: BHK-21 cell line (Cricetidae); BSR-T7 cell line (Animalia);

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NA cell line (Muridae); **rabies** virus (Rhabdoviridae (animal host only))--**neurotropic**, pathogen, recombinant, strain-CVS-B2c, strain-CVS-N2c, strain-SHBRV-18, strain-SN-10
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animal Viruses; Animals; Chordates; Mammals; Microorganisms; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates; Viruses
CHEMICALS & BIOCHEMICALS: glycoprotein; matrix protein; **rabies** virus G gene {**rabies** virus glycoprotein gene} (Rhabdoviridae (animal host only))
METHODS & EQUIPMENT: phenotypic analysis--analytical method; reverse genetics approach--analytical method, molecular genetic method
MISCELLANEOUS TERMS: reverse genetics; viral pathogenesis
CONCEPT CODES:
02506 Cytology and Cytochemistry-Animal
03502 Genetics and Cytogenetics-General
03506 Genetics and Cytogenetics-Animal
10064 Biochemical Studies-Proteins, Peptides and Amino Acids

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31500 Genetics of Bacteria and Viruses
33506 Virology-Animal Host Viruses
36006 Medical and Clinical Microbiology-Virology
BIOSYSTEMATIC CODES:
02624 Rhabdoviridae-animal host only (1993-)
33000 Animalia-Unspecified
86310 Cricetidae
86375 Muridae

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12678348 BIOSIS NO.: 200000431850
Spread and pathogenic characteristics of a G-deficient **rabies** virus recombinant: An in vitro and in vivo study.
AUTHOR: Etessami Reza; Conzelmann Karl-Klaus; Fadai-Ghotbi Babak; Natelson Benjamin; Tsiang Henri; Ceccaldi Pierre-Emmanuel(a)
AUTHOR ADDRESS: (a)Unit, Virology Department, Pasteur Institute, 25 rue du Dr Roux, 75724, Paris Cedex, 15**France
JOURNAL: Journal of General Virology 81 (9):p2147-2153 September, 2000
MEDIUM: print
ISSN: 0022-1317
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English
SUMMARY LANGUAGE: English

ABSTRACT: **Rabies** virus (RV), a highly **neurotropic** enveloped

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virus, is known to spread within the CNS by means of axonal transport. Although the envelope spike **glycoprotein** (**G**) of cell-free virions is required for attachment to neuronal receptors and for virus entry, its necessity for transsynaptic spread remains controversial. In this work, a G gene-deficient recombinant RV (SAD DELTAG) complemented phenotypically with RV **G protein** (SAD DELTAG+G) has been used to demonstrate the absolute requirement for G in virus transfer from one neuron to another, both in vitro, in neuronal cell cultures (cell line and primary cultures), and in vivo, in murine animal models. By using a model of stereotaxic inoculation into the rat striatum, infection is shown to be restricted to initially infected cells and not transferred to secondary neurons. In mouse as in rat models of infection, the limited infection did not cause any detectable symptoms, suggesting that G-deficient RV recombinants might be valuable as non-pathogenic, single-round vectors for expression of foreign genes.

DESCRIPTORS:

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MAJOR CONCEPTS: Infection
BIOSYSTEMATIC NAMES: Muridae--Rodentia, Mammalia, Vertebrata, Chordata, Animalia; Rhabdoviridae (animal host only)--Animal Viruses, Viruses, Microorganisms
ORGANISMS: mouse (Muridae)--animal model; **rabies** virus (Rhabdoviridae (animal host only))--G-deficient, pathogen, pathogenic characteristics, recombinant, spread, stereotaxic inoculation, strain-SAD delta-G; rat (Muridae)--animal model
ORGANISMS: PARTS ETC: CNS {central nervous system}--nervous system; secondary neurons--nervous system; striatum--nervous system
BIOSYSTEMATIC CLASSIFICATION (SUPER TAXA): Animal Viruses; Animals; Chordates; Mammals; Microorganisms; Nonhuman Mammals; Nonhuman Vertebrates; Rodents; Vertebrates; Viruses
DISEASES: **rabies** virus infection--nervous system disease, viral disease
CHEMICALS & BIOCHEMICALS: envelope spike glycoprotein
CONCEPT CODES:

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Display 3/9/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

36006 Medical and Clinical Microbiology-Virology
20504 Nervous System-Physiology and Biochemistry
20506 Nervous System-Pathology
33506 Virology-Animal Host Viruses
BIOSYSTEMATIC CODES:
02624 Rhabdoviridae-animal host only (1993-)

86375 Muridae

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Display 3/9/4 (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)

12527543 21376493 PMID: 11482987
Production and neurotropism of lentivirus vectors pseudotyped with lyssavirus envelope glycoproteins.
Desmaris N; Bosch A; Salaun C; Petit C; Prevost M C; Tordo N; Perrin P; Schwartz O; de Rocquigny H; Heard J M
Unite Retrovirus et Transfert Genetique, CNRS URA 1930, 28 Rue du Dr Roux, Paris, 75724, France.
Molecular therapy : the journal of the American Society of Gene Therapy (United States) Aug 2001, 4 (2) p149-56, ISSN 1525-0016
Journal Code: 100890581
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
We investigated the production efficiency and the gene transfer capacity

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DIALOG(R)File 154:MEDLINE(R)
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in the central nervous system of HIV-1-based vectors pseudotyped with either the **G protein** of the Mokola lyssaviruses (MK-G), a **neurotropic** virus causing **rabies** disease, or the vesiculo-stomatitis **G protein** (VSV-G). Both envelopes induced syncitia in cell cultures. They were incorporated into vector particles and mature virions were observed by electron microscopy. Vector production was two- to sixfold more efficient with VSV-G than with MK-G. For equivalent amounts of physical particles, vector titration was 5- to 25-fold higher with VSV-G than with MK-G pseudotypes on cultured cells, and in vivo gene expression in mouse brain was more intense. Thus, VSV-G pseudotypes were produced more efficiently and were more infectious than MK-G pseudotypes. Tropism for brain cells was analyzed by intrastriatal injections in rats. Both pseudotypes preferentially transduced neurons (70-90% of transduced cells). Retrograde axonal transport was investigated by instilling vector suspensions in the rat nasal cavity. Both pseudotypes were efficiently transported to olfactive neuron bodies. Thus, although coating HIV-1 particles with rabdhovirus envelope glycoproteins enables them to enter

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DIALOG(R)File 154:MEDLINE(R)
(c) format only 2003 The Dialog Corp. All rts. reserv.
neuronal cells efficiently, pseudotyping is not sufficient to confer the powerful neurotropism of lyssaviruses to lentivirus vectors. Copyright 2001 Academic Press.
Tags: Animal; Human; Support, Non-U.S. Gov't
Descriptors: *Brain--physiology--PH; *Genetic Vectors; *HIV-1--genetics --GE; *Lyssavirus--genetics--GE; *Viral Envelope Proteins--genetics--GE; Brain--cytology--CY; Brain--virology--VI; Cell Line; Corpus Striatum; Gene Transfer Techniques; Glucuronidase--genetics--GE; Glucuronidase --metabolism--ME; HIV-1--physiology--PH; Injections; Lyssavirus--physiology --PH; Mice; Microscopy, Fluorescence; Neurons--physiology--PH; Rats;

Transfection; Viral Envelope Proteins--metabolism--ME; beta-Galactosidase --genetics--GE; beta-Galactosidase--metabolism--ME

CAS Registry No.: 0 (G protein, vesicular stomatitis); 0 (Genetic Vectors); 0 (Viral Envelope Proteins)

Enzyme No.: EC 3.2.1.23 (beta-Galactosidase); EC 3.2.1.31 (Glucuronidase)

Record Date Created: 20010802

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Display 3/9/5 (Item 2 from file: 154)

DIALOG(R)File 154:MEDLINE(R)

09745291 98169512 PMID: 9501231

Rabies virus quasispecies: implications for pathogenesis.

Morimoto K; Hooper D C; Carbaugh H; Fu Z F; Koprowski H; Dietzschold B

Center for Neurovirology, Department of Microbiology and Immunology, Thomas Jefferson University, 1020 Locust Street, Philadelphia, PA 19107-6799, USA.

Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Mar 17 1998, 95 (6) p3152-6, ISSN 0027-8424

Journal Code: 7505876

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Passage of the mouse-adapted **rabies** virus strain CVS-24 (where CVS is challenge virus standard) in BHK cells results in the rapid selection of

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DIALOG(R)File 154:MEDLINE(R)

a dominant variant designated CVS-B2c that differs genotypically and phenotypically from the dominant variant CVS-N2c present in mouse-brain- or neuroblastoma-cell-passaged CVS-24. The glycoprotein of CVS-B2c has 10 amino acid substitutions compared with that of CVS-N2c. Because CVS-B2c can be reproducibly selected in BHK cells, it is likely to be a conserved minor subpopulation of CVS-24. CVS-N2c is more **neurotropic** in vitro and in vivo than CVS-B2c, which replicates more readily in nonneuronal cells in vitro and in vivo. These characteristics appear to be relevant to the pathogenicity of the two variants. CVS-N2c is more pathogenic for adult mice than CVS-B2c. In contrast, CVS-B2c is more pathogenic for neonatal mice. These differences in pathogenicity are reflected in the selection pattern when mixtures of CVS-N2c and CVS-B2c were used to infect neonatal and adult mice. Although CVS-N2c was highly selected in adult mice, no selection for either variant was seen in neonates, suggesting that certain aspects of development, such as maturation of the nervous and immune systems, may contribute to the selection process. We speculate that the existence of different variants within a **rabies** virus strain may

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DIALOG(R)File 154:MEDLINE(R)

facilitate the virus in overcoming barriers to its spread, both within the host and between species.

Tags: Animal; Comparative Study; Female

Descriptors: Glycoproteins--genetics--GE; ***Rabies**--etiology--ET; *

Rabies virus--genetics--GE; *Variation (Genetics); *Viral Envelope Proteins--genetics--GE; Age Factors; Amino Acid Sequence; Animals, Newborn; Brain--virology--VI; Evolution, Molecular; Genes, Viral; Hamsters; Kidney --virology--VI; Mice; Molecular Sequence Data; **Rabies**--mortality--MO; **Rabies** virus--classification--CL; **Rabies** virus--pathogenicity --PY; Sequence Analysis, DNA; Sequence Homology, Amino Acid; Virulence --genetics--GE

Molecular Sequence Databank No.: GENBANK/AF042823; GENBANK/AF042824

CAS Registry No.: 0 (Glycoproteins); 0 (Viral Envelope Proteins); 0 (glycoprotein G, rabies virus)

Record Date Created: 19980410

- end of record -

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Display 3/9/6 (Item 3 from file: 154)

DIALOG(R)File 154:MEDLINE(R)

09691809 98119772 PMID: 9451021

The role of site-specific N-glycosylation in secretion of soluble forms of **rabies** virus glycoprotein.

Wojczyk B S; Stwora-Wojczyk M; Shakin-Eshleman S; Wunner W H; Spitalnik S L

Department of Pathology and Laboratory Medicine, Wistar Institute, University of Pennsylvania, Philadelphia 19104, USA.

Glycobiology (ENGLAND) Feb 1998, 8 (2) p121-30, ISSN 0959-6658

Journal Code: 9104124

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Rabies virus glycoprotein is important in the biology and pathogenesis of **neurotropic** **rabies** virus infection. This

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DIALOG(R)File 154:MEDLINE(R)

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transmembrane glycoprotein is the only viral protein on the surface of virus particles, is the viral attachment protein that facilitates virus uptake by the infected cell, and is the target of the host humoral immune response to infection. The extracellular domain of this glycoprotein has N-glycosylation sequons at Asn37, Asn247, and Asn319. Appropriate glycosylation of these sequons is important in the expression of the glycoprotein. Soluble forms of **rabies** virus glycoprotein were constructed by insertion of a stop codon just external to the transmembrane domain. Using site-directed mutagenesis and expression in transfected eukaryotic cells, it was possible to compare the effects of site-specific glycosylation on the cell-surface expression and secretion of transmembrane and soluble forms, respectively, of the same glycoprotein. These studies yielded the surprising finding that although any of the three sequons permitted cell surface expression of full-length **rabies** virus glycoprotein, only the N-glycan at Asn319 permitted secretion of soluble **rabies** virus glycoprotein. Despite its biological and medical importance, it has not yet been possible to determine the crystal structure

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DIALOG(R)File 154:MEDLINE(R)

of the full-length transmembrane form of **rabies** virus glycoprotein which contains heterogeneous oligosaccharides. The current studies demonstrate that a soluble form of **rabies** virus glycoprotein containing only one sequon at Asn319 is efficiently secreted in the presence of the N-glycan processing inhibitor 1-deoxymannojirimycin. Thus, it is possible to purify a conformationally relevant form of **rabies** virus glycoprotein that contains only one N-glycan with a substantial reduction in its microheterogeneity. This form of the glycoprotein may be particularly useful for future studies aimed at elucidating the three-dimensional structure of this important glycoprotein.

Tags: Animal

Descriptors: Glycoproteins--metabolism--ME; *Glycoproteins--secretion --SE; ***Rabies** virus--physiology--PH; *Viral Envelope Proteins --metabolism--ME; *Viral Envelope Proteins--secretion--SE; Antigens, Viral --biosynthesis--BI; Antigens, Viral--immunology--IM; Antigens, Viral --metabolism--ME; CHO Cells; Carrier Proteins--metabolism--ME; Cell Line; Glycoproteins--genetics--GE; Glycoproteins--immunology--IM; Glycosylation

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Display 3/9/6 (Item 3 from file: 154)

DIALOG(R)File 154:MEDLINE(R)

--drug effects--DE; Hamsters; Indolizines--pharmacology--PD; Intracellular Fluid--metabolism--ME; Kinetics; Molecular Chaperones--metabolism--ME; Mutagenesis, Site-Directed; **Rabies** virus--genetics--GE; **Rabies** virus--immunology--IM; Recombinant Fusion Proteins--chemical synthesis--CS ; Recombinant Fusion Proteins--drug effects--DE; Recombinant Fusion Proteins--secretion--SE; Solubility; Swainsonine--pharmacology--PD; Transfection; Viral Envelope Proteins--genetics--GE; Viral Envelope Proteins--immunology--IM

CAS Registry No.: 0 (Antigens, Viral); 0 (Carrier Proteins); 0 (Glycoproteins); 0 (Indolizines); 0 (Molecular Chaperones); 0 (Recombinant Fusion Proteins); 0 (Viral Envelope Proteins); 0 (glycoprotein G, rabies virus); 0 (immunoglobulin heavy chain-binding protein); 72741-87-8 (Swainsonine); 79831-76-8 (castanospermine)

Record Date Created: 19980310

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Display 3/9/7 (Item 4 from file: 154)

DIALOG(R)File 154:MEDLINE(R)

07780769 93305903 PMID: 8318668

A model of the **rabies** virus glycoprotein active site.

Rustici M; Bracci L; Lozzi L; Neri P; Santucci A; Soldani P; Spreafico A; Niccolai N

Dipartimento di Biologia Molecolare Universita di Siena, Italia.

Biopolymers (UNITED STATES) Jun 1993, 33 (6) p961-9, ISSN 0006-3525

Journal Code: 0372525

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

The glycoprotein from the **neurotropic rabies** virus shows a significant homology with the alpha neurotoxin that binds to the nicotinic acetylcholine receptor. The crystal structure of the alpha neurotoxins suggests that the Arg 37 guanidinium group and the Asp 31 side-chain

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DIALOG(R)File 154:MEDLINE(R)

carboxylate of the erabutoxin have stereochemical features resembling those of acetylcholine. Conformational studies on the Asn194-Ser195-Arg196-Gly197 tetrapeptide, an essential part of the binding site of the **rabies** virus glycoprotein, indicate that the side chains of Asn and Arg could also mimic the acetylcholine structure. This observation is consistent with the recently proposed mechanism of the viral infection.

Tags: Comparative Study

Descriptors: *Glycoproteins--chemistry--CH; *Models, Molecular; *Viral Envelope Proteins--chemistry--CH; Amino Acid Sequence; Binding Sites; Computer Simulation; Molecular Sequence Data; **Rabies** virus; Sequence Homology, Amino Acid

CAS Registry No.: 0 (Glycoproteins); 0 (Viral Envelope Proteins); 0 (glycoprotein G, rabies virus)

Record Date Created: 19930730

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Display 3/9/8 (Item 1 from file: 98)
DIALOG(R)File 98:General Sci Abs/Full-Text

03544164 H.W. WILSON RECORD NUMBER: BGSI97044164 (THIS IS THE FULLTEXT)
Borna disease virus infection in animals and humans.
Richt, Jurgen A
Pfeuffer, Isolde; Christ, Matthias
Emerging Infectious Diseases (Emerging Infect Dis) v. 3 (July/Sept. '97) p. 343-52
SPECIAL FEATURES: bibl il ISSN: 1080-6040
LANGUAGE: English
COUNTRY OF PUBLICATION: United States
RECORD TYPE: Abstract; Fulltext RECORD STATUS: Corrected or revised record
WORD COUNT: 7096

ABSTRACT: The geographic distribution and host range of Borna disease (BD), a fatal neurologic disease of horses and sheep, are larger than previously thought. The etiologic agent, Borna disease virus (BDV), has

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DIALOG(R)File 98:General Sci Abs/Full-Text
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been identified as an enveloped nonsegmented negative-strand RNA virus with unique properties of replication. Data indicate a high degree of genetic stability of BDV in its natural host, the horse. Studies in the Lewis rat have shown that BDV replication does not directly influence vital functions; rather, the disease is caused by a virus-induced T-cell mediated immune reaction. Because antibodies reactive with BDV have been found in the sera of patients with neuropsychiatric disorders, this review examines the possible link between BDV and such disorders. Seroepidemiologic and cerebrospinal fluid investigations of psychiatric patients suggest a causal role of BDV infection in human psychiatric disorders. In diagnostically unselected psychiatric patients, the distribution of psychiatric disorders was found to be similar in BDV seropositive and seronegative patients. In addition, BDV-seropositive neurologic patients became ill with lymphocytic meningoencephalitis. In contrast to others, we found no evidence is reported for BDV RNA, BDV antigens, or infectious BDV in peripheral blood cells of psychiatric patients. Reprinted by permission of the publisher

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TEXT:

Borna disease (BD), first described more than 200 years ago in southern Germany as a fatal neurologic disease of horses and sheep, owes its name to the town Borna in Saxony, Germany, where a large number of horses died during an epidemic in 1885. The virus etiology of BD was proven in the early 1900s when Zwick and co-workers (1) in Giessen, Germany, successfully transmitted brain homogenates from infected horses to experimental animals. Other milestones in BD-related research were the demonstration of virus growth in cell cultures (2-4); the finding that the pathogenesis of BD is caused by a T-cell--dependent immune mechanism (5-8); and most recently, the molecular characterization of the etiologic agent of BD, the highly **neurotropic** Borna disease virus (BDV) (9-17).

BD is characterized by a disseminated nonpurulent meningoencephalomyelitis with infiltration of mononuclear cells (1,8,18,19) and a predilection for the gray matter of the cerebral hemispheres and the brain stem (8,19). In neurons, sometimes in glia cells, acidophilic intranuclear inclusion bodies, called Joest-Degen inclusion bodies, are

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occasionally found. BD occurs sporadically in Germany and Switzerland; its presence in other countries has not yet been substantiated. Natural infections in other Equidae, ruminants, rabbits, cats, and ostriches have also been described (19-21).

This review discusses the etiology of Borna disease, the natural and experimental infection in various animal species, the pathogenesis of the disease in the experimental rat model, the genetic stability of BDV, and the possible link between BDV or a similar agent and human neuropsychiatric disorders.

ETIOLOGY

The etiologic agent of BD, BDV, has been recently characterized as an enveloped, nonsegmented, negative-stranded RNA virus with a genomic size of approximately 9 kb and a nuclear site for replication and transcription (14-17). The genomic organization is similar to that of members of the Mononegavirales order; therefore, BDV is the prototype of the new family Bornaviridae within this order. The Mononegavirales also include

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DIALOG(R)File 98:General Sci Abs/Full-Text
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Filoviridae (e.g., Marburg and Ebola viruses), Paramyxoviridae (e.g., mumps, measles virus), and Rhabdoviridae (e.g., **rabies**, vesicular stomatitis virus). Six major open reading frames (ORFs)(I,II,III, IV,V,x1) are predicted in the genome sequence (16,17). Only five ORFs correspond to previously identified proteins with molecular weights of 10 (ORF x1: p10 BDV gene; 22), 18 (ORF III: gp18 BDV gene; 23), 24 (ORF II: p24 BDV gene; 24), 38/39 (ORF I: p38 BDV gene; 25), and 94 (ORF IV: p57 BDV gene; 26) or 84 (ORF IV: p57 BDV gene; 27, Richt et al., unpub. obs.) kDa.

NATURAL AND EXPERIMENTAL INFECTION

NATURAL INFECTION

Extensive epizootiologic studies in horses have shown that BD is rare but occurs all over Germany, extending beyond the classic disease-endemic regions (28-30). Furthermore, BDV-specific antibodies were detected in horses in several European countries, Israel (28,29), Japan (31), Iran (32), and the United States (33). Since BDV-specific antibodies are

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DIALOG(R)File 98:General Sci Abs/Full-Text

frequently found in clinically healthy horses (20,28,29), natural infections in horses seem to remain subclinical in most cases. Unknown exogenous and endogenous factors might influence the genesis of the disease (20).

In addition to its predominant natural host, the horse, other Equidae, sheep, cattle, rabbits, goats, deer, alpacas, llamas, cats, pigmy hippopotamus, sloth, vari monkeys (memur variegatus), and ostriches have become naturally infected with BDV (8,19,20,34,35). In sheep flocks, clinical BD can affect large numbers of animals; however, in horse stables, usually only a few animals show clinical signs. The virus is assumed to be transmitted through salival, nasal, or conjunctival secretions because BDV-specific RNA has been found in these secretions (20,28,29,36). Animals become infected by direct contact with these secretions or by exposure to contaminated food or water. A minimum incu-bation period of 4 weeks is estimated for horses and sheep with nonspecific signs such as hyperthermia, anorexia, colic, and constipation in the initial phase of the disease.

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During the acute phase of disease, neurologic signs such as ataxia, depression, circular movement, standing in awkward positions, collapsing, running into obstacles, and paralysis, result from nonpurulent meningoencephalomyelitis. Clinical illness usually lasts 1 to 3 weeks, and death rates for diseased horses are 80[percent] to 100[percent] (18-20,37). In surviving animals, recurrent episodes are possible, especially after stress (18). Clinical manifestations, however, may vary among individual animals and various species.

BD tends to occur in spring and early summer and is more frequent in some years than in others; therefore, arthropods have been discussed as a potential vector. BDV, however, has never been isolated from insects in Europe. In the Near East, ticks have been associated with transmission of an equine encephalomyelitis similar to BD (38). A definite virus reservoir for BDV has not been found; various rodents most likely represent such a reservoir. In addition, since many seropositive horses with subclinical infections have BDV-specific RNA in various secretions, they can be potential sources of infection for other animals and humans (29,36).

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DIALOG(R)File 98:General Sci Abs/Full-Text

EXPERIMENTAL INFECTION

BDV can be experimentally transmitted to a wide variety of animal species, from chickens to nonhuman primates (1,8,19,20,37). Incubation periods, clinical signs, and severity of the disease depend on the animal species and the virus variants. Rabbits, Lewis rats, and guinea pigs are highly

susceptible, whereas chickens, monkeys, cattle, and tree shrews (Tupaia glis;39) are less susceptible (8,19). Some animals (hamsters, black-hooded rats, mice, ferrets, pigeons, and dogs), however, do not develop disease despite being persistently infected with BDV (1,8,18,19). The pathohistologic picture after experimental infections resembles that of natural infections. Most experimentally infected animals develop perivascular and parenchymal central nervous system (CNS) infiltrations and have infectious virus in the brain tissue. Frequently, the clinical picture in experimentally infected animals does not differ from that of spontaneously infected natural hosts.

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DIALOG(R)File 98:General Sci Abs/Full-Text

The result of BDV infection in rats depends on the inbred rat strain and the virus isolate used for infection. Black-hooded rats do not show clinical signs after BDV infection with BDV isolates despite persistent virus infection and mononuclear infiltration of the CNS (40). Lewis rats, however, are highly susceptible and are therefore used extensively for studies of BD pathogenesis (see below; 5,8,19). When infected with various BDV variants, Lewis rats exhibited clinical manifestations such as behavioral disorders, paralytic disease, or obesity, in addition to fertility problems (8,19), which indicates that BDV can form virus variants with different biologic properties.

PATHOGENESIS

BDV is a highly **neurotropic** agent that gains access to the CNS, probably by intraaxonal migration through the olfactory nerve or nerve endings in the oropharyngeal and intestinal regions (18,41). Virus spreads throughout the CNS by intraaxonal transport and centrifugally into the peripheral nerves. Antibody titers in naturally infected animals, such as

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DIALOG(R)File 98:General Sci Abs/Full-Text

in horses with clinical BD, are relatively low and are found in sera and cerebrospinal fluid (CSF) (19,20,28,29). Whether BDV-specific antibodies are neutralizing is not certain (5,42); the coexistence of BDV-specific antibodies and infectious virus in the CSF contradicts this assumption (43).

Extensive studies in the Lewis rat showed that the immune response to viral antigens after BDV infection does not elicit protective immunity but rather an immunopathologic reaction in which T cells play an important role (5,44). After adult rats are infected intracerebrally or intranasally, productive virus replication is found in the entire CNS. Once introduced into the rat's CNS, BDV usually causes a persistent infection with continuous productive replication in the brain and spinal cord (5,8). In immunocompetent animals, no infectivity was found in extraneural tissues at any stage of infection (5,45). In newborn animals, in contrast, the virus spreads throughout the whole organism; BDV-specific antigen was found in parenchymal cells of numerous organs, and infectious virus was found in

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Display 3/9/9 (Item 1 from file: 266)
DIALOG(R)File 266:FEDRIP

00299540
IDENTIFYING NO.: 5R01AI45097-03 AGENCY CODE: CRISP
MOLECULAR PATHOGENESIS OF **RABIES**
PRINCIPAL INVESTIGATOR: DIETZCHOLD, BERNHARD
ADDRESS: THOMAS JEFFERSON UNIVERSITY 1020 LOCUST STREET PHILADELPHIA, PA 19107
PERFORMING ORG.: THOMAS JEFFERSON UNIVERSITY, PHILADELPHIA, PENNSYLVANIA
SPONSORING ORG.: NATIONAL INSTITUTE OF ALLERGY AND INFECTIOUS DISEASES
FY : 2001 TYPE OF AWARD: Noncompeting Continuation (Type 5)
SUMMARY: To date, the molecular mechanisms involved in the pathogenesis of either canine street **rabies** or the newly emerging infections with the silver- haired bat-associated **rabies** virus strain (SHBRV) remain obscure. The purpose of this proposal is to delineate these mechanisms with particular focus on interactions between **rabies** virus and neurons. Our preliminary data reveal that tissue cultured-adapted, mouse brain-adapted and natural street **rabies** virus strains, which differ greatly

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DIALOG(R)File 266:FEDRIP

in their pathogenicity in vivo, also differ markedly in the expression levels of the **rabies** virus structural proteins, in particular the **glycoprotein** (**G**), in cultured neurons. Surprisingly, the pathogenicity index of a particular **rabies** virus strain correlates inversely with the expression of the viral **G protein** on the surface of the infected neuron. One possibility is that this results in a self-limiting infection by preventing the transport of the **rabies** virus nucleoprotein complex into the periphery of the neuron, i.e., into neuronal processes, and hence axonal spread of the virus. In infections with highly pathogenic **rabies** virus strains such as SHBRV, the expression of the **G protein** in neurons is minimal and apoptosis does not occur until the infection has spread to the next uninfected neuron. We intend to investigate the mechanism(s) involved in the regulation of viral gene expression in neurons and identify proteins associated with the pathogenicity of street **rabies** virus strains in order to better understand the pathogenesis of human **rabies** , especially the cryptic human **rabies** cases caused by SHBRV that have

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DIALOG(R)File 266:FEDRIP
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occurred recently in North America.
DESCRIPTORS: laboratory mouse; genetic strain; polymerase chain reaction; molecular pathology; gene expression; virus genetics; immunoprecipitation; virulence; **rabies**; neuron; neuronal transport; messenger RNA; protein sequence; glycoprotein; ribonucleoprotein; virus protein; proteolysis; tissue /cell culture; **neurotropic** virus; **rabies** virus; apoptosis; neuropathology; neuroregulation; proteasome; emerging infectious disease

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? rabies (10n) neurotropic
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44585 RABIES
15172 NEUROTROPIC

S4 132 RABIES (10N) NEUROTROPIC
? rd s4
...examined 50 records (50)
...examined 50 records (100)
...completed examining records
S5 41 RD S4 (unique items)
? d s5/3/1-41
Display 5/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13618263 BIOSIS NO.: 200200247084
Interaction of the poliovirus receptor CD155 with the dynein light chain Tctex-1 and its implication for poliovirus pathogenesis.
AUTHOR: Mueller Steffen; Cao Xuemei; Welker Reinhold; Wimmer Eckard(a)
AUTHOR ADDRESS: (a)Dept. of Molecular Genetics and Microbiology, State University of New York, Stony Brook, NY, 11794**USA E-Mail: ewimmer@ms.cc.sunysb.ed
JOURNAL: Journal of Biological Chemistry 277 (10):p7897-7904 March 8, 2002
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

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Display 5/3/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

13368892 BIOSIS NO.: 200100576041
Extensive attenuation of rabies virus by simultaneously modifying the dynein light chain binding site in the P protein and replacing Arg333 in the G protein.
AUTHOR: Mebatsion Teshome(a)
AUTHOR ADDRESS: (a)Department of Virology, Intervet International B.V., 5830 AA, Boxmeer: teshome.mebatsion@intervet.com**Netherlands
JOURNAL: Journal of Virology 75 (23):p11496-11502 December, 2001
MEDIUM: print
ISSN: 0022-538X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

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Ref    Items  Index-term
E1         2  AU=MITRAOUI A
E2         2  AU=MITRAPHANOUS K A
E3         0 *AU=MITRAPHANOUS KYRLACOS
E4         1  AU=MITRAPHANOUS, KYRIACOS ANDREOU
E5         1  AU=MITRAS, JOAO LUIS
E6         3  AU=MITRASCA, B.
E7         1  AU=MITRASCA, BRADUT
E8         5  AU=MITRASCA, D.
E9         4  AU=MITRASCA, DANA
E10        1  AU=MITRASCA, E.
E11        7  AU=MITRASENA, U.
E12        9  AU=MITRASETIA T

           Enter P or PAGE for more
? ellard fiona

Ref    Items  Index-term
E1         0 *LLARD FIONA
E2         1  LLARDECANS
E3         1  LLARDERELLO
E4         3  LLARENA
E5         3  LLARENAI
E6         1  LLARES
E7        12  LLARETA
E8         6  LLARG
E9         6  LLARGA
E10        5  LLARGE
E11        1  LLARGER
E12        7  LLARGUETA

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? e au=ellard fiona

Ref    Items  Index-term
E1        30  AU=ELLARD F M
E2         9  AU=ELLARD F.M.
E3         0 *AU=ELLARD FIONA
E4        13  AU=ELLARD FIONA M
E5         9  AU=ELLARD FM
E6         2  AU=ELLARD G
E7       241  AU=ELLARD G A
E8         1  AU=ELLARD G E
E9         2  AU=ELLARD G.
E10       86  AU=ELLARD G.A.
E11       75  AU=ELLARD GA
E12        9  AU=ELLARD GORDON A

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? e au=ellard, fiona m

Ref    Items  Index-term
E1         2 *AU=ELLARD, FIONA M
E2         8  AU=ELLARD, FIONA M.
E3         5  AU=ELLARD, FIONA MARGARET
E4         1  AU=ELLARD, G.
E5        30  AU=ELLARD, G. A.
E6         1  AU=ELLARD, GEORGE
E7         7  AU=ELLARD, GORDON A.
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E8	1	AU=ELLARD, GREGG
E9	1	AU=ELLARD, GREGORY
E10	2	AU=ELLARD, GREGORY A.
E11	2	AU=ELLARD, H.
E12	5	AU=ELLARD, J.

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